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Evaluation of Some Inflammatory and Biochemical Markers in Obese and Lean Iraqi Women

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Abstract

The present study aims to detecting several biochemical markers and cytokines in obese and normal lean Iraqi women. Forty women (٢٠ obese and ٢٠ lean) were chosen from different areas in Baghdad city there ages ranges between ٢٠-٣٧ years and body mass index (BMI) between ٢٠,٧٥-٣٥,٦ Kg/m². The study showed a high significant increment in Fasting Blood Glucose (FBG), serum cholesterol and Homeostasis Model Assessment (HOMA) in obese women as compared with lean and a significant increment in HDL (High Density Lipoprotein) in lean women when compared with obese ones. A significant correlation coefficient (r) was noticed between BMI and studied parameters, FBG, cholesterol, triglyceride, insulin, HOMA, high sensitive C Reactive Protein
(hs-CRP), Tumor Necrosis Factor alfa (TNF-α) and IL-٦.

Keywords: Obesity. BMI, Cholesterol, Inflammatory Mediator.

Introduction

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems [١,٢] Obesity increases the likelihood of various diseases, particularly heart disease, type ٢ diabetes, obstructive sleep apnea, certain types of cancer, osteoarthritis [٢] and asthma [٣]. It is most commonly caused by a combination of excessive food energy intake, lack of physical activity, and genetic susceptibility, although a few cases are caused primarily by genes. Obesity is defined by body mass index (BMI) and further evaluated in terms of fat distribution via the waist–hip ratio and total cardiovascular risk factor [٤,٥]. BMI is closely related to both percentage body fat and total body fat [٦]. As a result, obesity has been found to reduce life expectancy [٢]. At an individual level, a combination of excessive food energy intake and a lack of physical activity is thought to explain most cases of obesity [٧]. A limited number of cases are due primarily to genetics, medical reasons, or psychiatric illness[٨].In contrast, increasing rates of obesity at a societal level are felt to be due to an easily accessible and palatable diet [٩] increased reliance on cars, and mechanized manufacturing [١٠,١١]. Obesity is one of the leading preventable causes of death world wide Large scale [٨,١٢,١٣]. American and European studies have found that mortality risk is lowest at a BMI of ٢٠–٢٥ kg/m^{٢,} in non-smokers [١٤,١٥] and at ٢٤-٢٧ kg/m^{٢} in current smokers, with risk increasing along with changes in either direction. [١٦,١٧]. A BMI above ٣٢ kg/m^{٢} has been associated with a doubled mortality rate among women over a ١٦-year period[١٨]. In the United States obesity is estimated to cause ١١١,٩٠٩ to ٣٦٥,٠٠٠ deaths per year [٢,١٣], while ١ million (٧,٧٪) of deaths in Europe are attributed to excess weight[١٩,٢٠]. On average, obesity reduces life expectancy by six to seven years [٢,٢١]. BMI of ٣٠–٣٥ kg/m^{٢} reduces life expectancy by two to four years [١٥], while severe obesity (BMI > ٤٠ kg/m^{٢}) reduces life expectancy by ten years [١٥]. Complications are either directly caused by obesity or indirectly related through mechanisms sharing a common cause such as a poor diet or a sedentary lifestyle. The strength of the link between obesity and specific conditions varies. One of the strongest is the link with type ٢ diabetes. Excess body fat underlies ٦٤٪ of cases of diabetes in men and ٧٧٪ of cases in women [٢٢]. Health consequences fall into two broad categories: those attributable to the effects of increased fat mass (such as osteoarthritis, obstructive sleep

apnea, social stigmatization) and those due to the increased number of fat cells (diabetes, cancer, cardiovascular disease, non-alcoholic fatty liver disease) [٢,٢٣]. Increases in body fat alter the body's response to insulin, potentially leading to insulin resistance. Increased fat also creates a proinflammatorystate [٢٤,٢٥] and a prothrombotic state[٢٣,٢٦]. This studied aimed to matured some inflammatory and Biochemical parameter and compared results between lean and obese women.

Experimental Work

Patients

The study groups included forty women ٢٠ obeseand ٢٠ lean control. Women were chosen randomly according to their weight, their ages ranged ٢٠-٣٧ years, The controls were selected among subjects who were healthy in terms of regular cycle, normal hormonal assay, non-diabetic, non-hypertensive, no other endocrine disorders and were free of acute illness or infection at time of sampling. In all cases, body weight and height, BMI were measured by using standard methods. All cases were investigated for the Serum levels of insulin, cytokines (IL-٦ and TNF-α), high sensitive C-Reactive protein in addition to plasma level of glucose and Lipid profile From each cases, ١٠ ml of blood were obtained by venepuncture, using a ١٠ ml disposable syringe between ٩,٠٠ and ١١,٠٠ A.M. The blood sample was divided into two aliquots; ٣ and ٧ml. the first aliquot was used for the estimation of plasma glucose and lipid profile. The second aliquot was dispensed in a plain tube and left for an hour to clot at room temperature ٢٢°C, and then centrifuged at ٣٠٠٠ rpm for ١٠ minutes to collect serum. The serum was divided into aliquots (٢٥٠µl) in Eppendorff tubes and stored in the freezer at -٢٠ºC until use.

Measurements of Markers

- Serum TNF-α concentration was determined by the sandwich ELISA method (Phoenix Pharmaceuticals, INC, USA).
- Serum IL-٦ concentration was determined by the competitive ELISA method (Raybiotech Company, USA).
- Serum hs-CRP concentration was determined by the competitive ELISA method (Demedetec Company, Germany).
- Serum insulin concentration was determined by the sandwich ELISA method Serum (Demedetec Company, Germany).
- Cholesterol level was measured by enzymatic end point method supplied by (Bio Labo Company, Franc).
- Triglyceride was determined after enzymatic hydrolysis with lipase into fatty acids and glycerol. The resultant glycerol is then phosphorelatedin the presence of ATP and glycerokinase to give hydrogen peroxide that react in presence of peroxidase with ٤-aminoantipyrine and parachlorophenol to give colored chromophen equinoneimie (Bio Labo Company, Franc).
- Low-density lipoprotein-cholesterol was estimated by using formula of Friedwald [٢٧].

  LDL - cholesterol = Total cholesterol – [HDL-cholesterol + TG/٥]
- Very low-density lipoprotein- cholesterol was estimated by using formula of Friedwald [٢٧].

  VLDL-Cholesterol = TG/٥
- Insulin resistance (IR) was determined by a number of different methods including fasting insulin, glucose for calculation (insulin measured µU/ml, glucose measured in mg/dl), the homeostasis model assessment (HOMA). The estimation of insulin resistance by HOMA score was calculated using Matthews formula [٢٨].

  HOMA = [Fasting serum insulin (µU/ml) × Fasting blood glucose (mmol/L)] / ٢٢,٥

Statistical Analysis

Results were analysed using Statistical Analysis System- SAS (٢٠١٠) T-test was used to significant compared between means. Correlation coefficient was estimated.

Results and Discussion

In the present study, groups were divided in two dependent on BMI obese and lean to note the effect of obesity on the studied factors.

Table (١)
Inflammatory and biochemical markers in Obese and Lean Iraqi women.

Parameters studied	*Lean*	*Obese*	*Level of Sig.*
TNF- α	٢,٢٣±٠,٨٣	٢,٧٢±١,٠٧	NS
IL-٦	١١,٧٢±٦,٩٧	١٥,٣٩±٦,٠٥	NS
hs-CRP	١,٨٩±١,٧٥	٢,٧٣±١,١٩	NS
Fasting Serum Insulin	١٢,٦٠±١,٠٨	١٤,٥٥ ± ٣,٣٥	NS
Triglyceride	١١١,٥٥±١٨,١٢	١٢٢,٧٠±١٩,٧٩	NS
HDL	٦٥,٢٥±٩,١٧	٥٨,٣٥±١٣,٢٣	*
VLDL	٢٢,٩٤±٤,١١	٢٣,١٧±٤,٣٤	NS
Serum Cholesterol	١٧٠,٧٠±١١,٦٤	١٨٦,١٠±١٠,٤٧	**
FBG	٧٧,٧٠± ٠,٠٧	٨٩,٧٥±٣٠,٠٠	**
HOMA	٤٣,٣٧±١٨,٠١	٥٧,٨٧±١٣,١٠	**

**** (P≤٠,٠٥), ** (P≤٠,٠١), NS: Non-significant.***

The mean serum level of Tumor Necrosis Factor-α (TNF-α) was non significantly elevation between lean (٢,٢٣±٠,٨٣ pg/ml) and obese once (٢,٧٢±١,٠٧pg/ml), in serum Interleukin- ٦ (IL-٦) the mean values (١١,٧٢±٦,٩٧pg/ml) for lean and (١٥,٣٩±٦,٠٥ pg/ml) for obese found to be non-significantly elevated, it was also for Serum High sensitive C-Reactive Protein (hs-CRP) found to be non-significantly elevated in obese (٢,٧٣±١,١٩ mg/L) witha mean of (١,٨٩±١,٧٥mg/L) in lean women**,** Fasting serum insulin showed non-significant results between lean (١٢,٦٠±١,٠٨μU/ml) and obese once (١٤,٥٥±٣,٣٥μU/ml), in Triglyceride there was no significant in lean women (١١١,٥٥±١٨,١٢mg/١٠٠ml) when it compared with obese once (١٢٢,٧٠±١٩,٧٩mg/١٠٠ml), in High Density Lipoprotein (HDL) there was a significant increase in lean women (٦٥,٢٥±٩,١٧ml/dl) when compared with obese once (٥٨,٣٥±١٣,٢٣ml/dl), there was no significant increase in Very Low Density Lipoprotein (VLDL) between lean women (٢٢,٩٤±٤,١١mg/dl) when it compared with obese once (٢٣,١٧±٤,٣٤mg/dl), there was a significant increments in serum cholesterol in obese women (١٨٦,١٠±١٠,٤٧mg/١٠٠ml) when it compared with lean once (١٧٠,٧٠±١١,٦٤), also there was a significant increments in Fasting Blood Glucose (FBG) in obese women (٨٩,٧٥±٣٠,٠٠mg/dl) when it compared with lean once, and there was a significant increment in Homeostasis Model Assessment (HOMA) in obese women (٥٧,٨٧±١٣,١٠) when it compared with lean once(٤٣,٣٧± ٨,٠١).

Correlation coefficient (r) between BMI and studied parameters

A significant correlation was found between BMI among FBG, Cholesterol, Triglyceride, HOMA, insulin, hs-CRP, TNF-α, IL-٦ as shown in Table (٢).

Table (٢)
Correlation coefficient (r) between BMI and studied parameters.

Parameters studied with BMI	*Correlation coefficient*	*Level. of sig.*
FBG	٠,٤٢	**
Cholesterol	٠,٥٧	**
Triglyceride	٠,٣٣	*
HDL	- ٠,٢٤	NS
VLDL	٠,٠٤	NS
Insulin	٠,٢٨	*
HOMA	٠,٤١	**
hs-CRP	٠,٤١	**
TNF-α	٠,٤٢	**
IL-٦	٠,٤٠	**

**** (P≤٠,٠٥), ** (P≤٠,٠١), NS: Non-significant***

The concentration of HDL-cholesterol is adversely altered in obesity, with HDL-cholesterol levels associated with both the degree and distribution of obesity. More specifically, intra - Abdominal visceral fat deposition is an important negative correlate of HDL-cholesterol. The specific subfractions of HDL that are altered in obese states include the HDL٢, apolipoprotein A-I, and pre-beta١ subfractions. Decreased HDL levels in obesity have been attributed to both an enhancement in the uptake of HDL٢ by adipocytes and an increase in the catabolism of apolipoprotein A-I on HDL particles. In addition, there is a decrease in the conversion of the pre-beta١ subfraction, the initial acceptor of cholesterol from peripheral cells, to pre-beta٢ particles [٢٩]. Obesity is at epidemic levels in all age groups. The effect of obesity on cholesterol levels is complex. Overweight individuals

tend to have high triglyceride and LDL levels and low HDL levels. This combination is a risk factor for heart disease, Obesity also causes other effects (high blood pressure, increase in inflammation) that pose major risks to the heart. Darvall and his research team reported that increased levels of both triglycerides and free fatty acids are associated with obesity and insulin resistance [٣٠].

Obesity is particularly dangerous when it is one of the components of the metabolic syndrome, formerly known as syndrome X. This syndrome consists of obesity marked by abdominal fat, unhealthy cholesterol levels, high blood pressure, and insulin resistance. Metabolic syndrome is a pre-diabetic condition that is significantly associated with heart disease and higher mortality rates from all causes. Many doctors recommend that patients with metabolic syndrome should be aggressively treated with high-dose statin therapy to lower LDL levels. Obesity is also strongly associated with type ٢ diabetes, which itself poses a significant risk for high cholesterol levels and heart disease [٣٠].

A higher BMI is associated with higher homeostasis model assessment values for insulin resistance (HOMA-IR), homeostasis model assessment of β-cell function (HOMA-β), and insulinogenic index as well as lower levels of insulin sensitivity index composite (ISI_{comp}) and disposition index (DI) [٣١].

These changes are consistent with the lipid profile that is typically found in association with insulin resistance. The effects of insulin resistance on lipid metabolism are well known. Increased secretion of very low-density lipoprotein (VLDL) particles by the liver results in elevated plasma TG concentrations. Subsequently, TGs are exchanged for cholesteryl ester (CE) by the activity of CE transfer protein. This process results in TG-enriched high-density lipoprotein (HDL) particles that are catabolized more rapidly, and CE-enriched VLDL particles that are converted into small dense low-density lipoprotein (LDL) particls [٣٢]. As a consequence, insulin resistance contributes to decreased plasma levels of HDL-C and apolipoprotein (apo) A-I, and higher levels of apo B [٣٣].

The HOMA is a method for assessing ß-cell function and IR from fasting glucose and insulin or C-peptide concentrations. The relationship between glucose and insulin in the basal state reflects the balance between hepatic glucose output and insulin secretion, which is maintained by a feedback loop between the liver and ß-cells [٣٤]. Decreases in ß-cell function were modeled by changing the ß-cell response to plasma glucose concentrations. Insulin sensitivity was modeled by proportionately decreasing the effect of plasma insulin concentrations at both the liver and the periphery [٣٤]. Some investigators had recommended calculating an index of IR from glucose and insulin levels (e.g. HOMA) [٣٥]. In previous study HOMA was considered as valid method to assess insulin sensitivity in epidemiological studies [٣٦].

Cytokines such as TNF-α influence several metabolic activities, including glucose and lipid metabolism [٣٧]. Since obesity and IR are frequent findings in hyper androgenic women in the current study, TNF-α concentration were positively correlated with BMI, as shown in table ٢. Previous studies have shown the relationship between BMI and TNF-α level [٣٨,٣٩].

The secretion of IL-٦ is regulated by several physiologic or pathologic factors: hormones, cytokines, diet, physical activity, stress, hypoxia, and others. Adipose tissue-derived IL-٦ may have an effect on metabolism through several mechanisms, including adipose tissue-specific gene expression, triglyceride release, lipoprotein lipase down regulation, insulin sensitivity, and so on [٤٠].

IL-٦ is believed to be beneficial for insulin-regulated glucose metabolism in muscle. Furthermore, the effects of the cytokine are seemingly influenced by whether it is present acutely or chronically; the latter is the setting associated with insulin resistance [٤١].

Conclusion

In the present study, there was a significant increment in Fasting Blood Glucose (FBG), serum cholesterol and Homeostasis Model Assessment (HOMA) in obese womens as compared with lean once

and a significant increment in HDL (High Density Lipoprotein) in lean womens when compared with obese once. A significant correlation coefficient (r) was noticed between BMI and studied parameters, FBG, cholesterol, triglyceride, insulin, HOMA, high sensitive C Reactive Protein (hs-CRP), Tumor Necrosis Factor alfa (TNF-α) and IL-٦.

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الخلاصة

استهدفت الدراسة تحديد تراكيز بعض الهرمونات والسايتوكينات المرتبطة بالنساء البدينات ومقارنتها مع مجموعة النساء النحيفات الطبيعيات تم اختيار (٤٠) امراة (٢٠ بدينات و٢٠ نحيفات) من اماكن مختلفة في مدينة بغداد تراوحت اعمارهن ما بين (٢٠–٣٧) سنة و تراوح مؤشر او معامل كتلة الجسم بين (٢٠,٧٥–٣٥,٦) كغم/م^{٢} اظهرت النتائج وجود زيادة معنوية في الكلوكوز الصيامي والكولسترول و HOMA عند النساء البدينات بالمقارنة مع النساء النحيفات وان هناك زيادة معنوية في مستوى اللزوجة العالية للبروتين الدهني للنساء النحيفات عند مقارنتها مع النساء البدينات وان هناك علاقة طردية بين مؤشر او معامل كتلة الجسم مع الكلوكوز الصيامي والكولسترول والكلسريد

الثلاثي والانسولين وHOMA والبروتين المرتبط سي عالي الحساسية ومعدل النخر الهرمي والانترلوكين ٦.